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(FILE 'HOME' ENTERED AT 15:23:28 ON 13 AUG 2007)

FILE 'BIOSIS, CAPLUS, EMBASE, MEDLINE, JAPIO' ENTERED AT 15:23:46 ON 13 AUG 2007

L1	1924	S	(ANTI CD4 ANTIBOD?)
L2	839	S	(ANTI CD40 ANTIBOD?)
L3	3	S	L1 AND L2
L4	3		DUPLICATE REMOVE L3 (0 DUPLICATES REMOVED)
L5	0	S	(CDLOCD40HI)
L6	6661	S	CD4 AND CD40
L7	2554	S	L6 AND ANTIBOD?
L8	1638	S	L7 AND PD<2004
L9	876		DUPLICATE REMOVE L8 (762 DUPLICATES REMOVED)
L10	34	S	L9 AND DIABETES?
L11	1	S	L9 AND EMPHYSEMA?
L12	803		DUPLICATE REMOVE L1 (1121 DUPLICATES REMOVED)
L13	661	S	L12 AND PD<2004
L14	7	S	L9 AND L1
L15	25	S	L9 AND L2

L15 ANSWER 11 OF 25 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN
AN 2000:432374 BIOSIS
DN PREV200000432374
TI Increased expression of CD40 ligand in activated CD4+ T lymphocytes of systemic sclerosis patients.
AU Valentini, Gabriele [Reprint author]; Romano, Maria Fiammetta; Naclerio, Caterina; Bisogni, Rita; Lamberti, Annalisa; Turco, Maria Caterina; Venuta, Salvatore
CS Istituto di Clinica Medica e Reumatologia, II Universita di Napoli, Via Pansini, 5, 80131, Napoli, Italy
SO Journal of Autoimmunity, (August, 2000) Vol. 15, No. 1, pp. 61-66. print.
ISSN: 0896-8411.
DT Article
LA English
ED Entered STN: 11 Oct 2000
Last Updated on STN: 10 Jan 2002
AB CD40-CD154 interactions play a key role in regulating immune response and are involved in the development of some autoimmune diseases. We analysed the expression of CD154 antigen in CD3-activated PBMC from 10 systemic sclerosis (SSc) patients and 10 control subjects by immunofluorescence. PBMC from SSc patients showed an increased expression of this molecule, since, 6 h following CD3 stimulation, the percentage of CD154+ cells was of 17.53+-2.0 (mean+-SE) in control and 25.33+-2.93 in patient cells ($P<0.03$). The higher expression of CD154 antigen was ascribible to CD4+ cells. The enhanced induction of CD154 following CD3 stimulation depended on protein synthesis, since was abolished when the cells were stimulated via CD3 in the presence of cycloheximide. By analysing the expression of the CD40-induced antigen CD80, we verified that a blocking anti-CD40 antibody inhibited CD80 appearance in SSc activated monocytes, indicating that CD154 molecule was functional. These results show an enhanced expression of a functional CD154 molecule in SSc CD4+ activated T lymphocytes.
CC Cytology - Human 02508
Cytology - Animal 02506
Biochemistry studies - General 10060
Biochemistry studies - Proteins, peptides and amino acids 10064
Blood - Blood and lymph studies 15002
Blood - Blood cell studies 15004
Bones, joints, fasciae, connective and adipose tissue - Pathology 18006
Immunology - General and methods 34502
Immunology - Immunopathology, tissue immunology 34508
IT Major Concepts
Biochemistry and Molecular Biophysics; Clinical Immunology (Human Medicine, Medical Sciences)
IT Parts, Structures, & Systems of Organisms
CD154-positive cells: blood and lymphatics, immune system; CD4 -positive T lymphocytes: blood and lymphatics, immune system; PBMC: blood and lymphatics, immune system, CD3-activated
IT Diseases
systemic sclerosis: connective tissue disease
Sclerosis (MeSH)
IT Chemicals & Biochemicals
CD154: expression, functionality; CD3; CD80: CD40-induced antigen, expression; anti-CD40 antibody; cycloheximide: protein synthesis inhibitor; protein: synthesis
ORGN Classifier
Hominidae 86215
Super Taxa
Primates; Mammalia; Vertebrata; Chordata; Animalia
Organism Name

human: patient
Taxa Notes
Animals, Chordates, Humans, Mammals, Primates, Vertebrates
RN 66-81-9 (cycloheximide)

ANSWER 1 OF 7 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN
AN 1995:438519 BIOSIS
DN PREV199598452819
TI Characteristics of antigen-independent and antigen-dependent interaction of dendritic cells with CD4+ T cells.
AU Hauss, Pascale; Selz, Francoise; Cavazzana-Calvo, Marina; Fischer, Alain [Reprint author]
CS INSERM U429, Hop. Necker-Enfants Malades, 149 rue de Sevres, F-75743 Paris Cedex 15, France
SO European Journal of Immunology, (1995) Vol. 25, No. 8, pp. 2285-2294.
CODEN: EJIMAF. ISSN: 0014-2980.
DT Article
LA English
ED Entered STN: 10 Oct 1995
Last Updated on STN: 10 Oct 1995
AB Dendritic cells (DC) are the main antigen-presenting cells for the initiation of primary T cell-mediated immune responses. In the first stage of activation, T cells bind to DC in an antigen-independent manner. We studied the adhesion characteristics of human CD4+ T cells to DC generated from CD34+ hematopoietic progenitors following 12 to 13 days of culture in the presence of granulocyte/macrophage colony-stimulating factor and tumor necrosis factor-alpha. A majority of these cells had the morphology, phenotype and functions of DC. CD4+ T/DC adhesion was measured by means of fluorescence microscopy and flow cytometry. Four independent receptor/ligand pathways, LFA-1/ICAM, ICAM/LFA-1, CD2/LFA-3 and CD28/CD80, were involved in the transient adhesion of DC to CD4+ T cells in antigen-independent and specific alloantigen-dependent situations, as shown by blocking experiments using monoclonal antibodies. The antibodies also blocked a primary mixed lymphocyte reaction (MLR) in which DC were used as stimulatory cells. Adhesion of alloreactive CD4+ T cells to antigen-presenting DC was stronger than that of resting CD4+ T cells, while peak adhesion occurred after 5 and 20 min, respectively. The LFA-1 ligands involved in adhesion of resting CD4 T cells to DC and alloreactive CD4+ T-cells to specific DC differed in part, since ICAM-3 on resting T cells and ICAM-1 on alloreactive T lymphocytes preferentially bound LFA-1. Studies of interactions between DC and phorbol ester-activated T cells expressing the CD40 ligand revealed a fifth independent adhesion pathway, CD40/CD40 ligand. CD4-mediated regulation of CD4+ T/DC adhesion was suggested by the observation that preincubation of CD4+ T cells and DC individually with anti-CD4 antibodies inhibited adhesion. In addition, antibodies specific for HLA class II molecules inhibited adhesion when used to pretreat DC but not alloactivated CD4+ T cells.
CC Cytology - Human 02508
Genetics - Human 03508
Biochemistry methods - Proteins, peptides and amino acids 10054
Biochemistry methods - Carbohydrates 10058
Biochemistry studies - Proteins, peptides and amino acids 10064
Biochemistry studies - Carbohydrates 10068
Biophysics - Methods and techniques 10504
Biophysics - Molecular properties and macromolecules 10506
Biophysics - Membrane phenomena 10508
Blood - Blood cell studies 15004
Blood - Lymphatic tissue and reticuloendothelial system 15008
Bones, joints, fasciae, connective and adipose tissue - Anatomy 18002
Integumentary system - Anatomy 18502
Development and Embryology - Morphogenesis 25508
In vitro cellular and subcellular studies 32600
Immunology - Immunopathology, tissue immunology 34508
IT Major Concepts
Biochemistry and Molecular Biophysics; Blood and Lymphatics (Transport

ANSWER 1 OF 7 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN
AN 1995:438519 BIOSIS
DN PREV199598452819
TI Characteristics of antigen-independent and antigen-dependent interaction of dendritic cells with CD4+ T cells.
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Genetics - Human 03508
Biochemistry methods - Proteins, peptides and amino acids 10054
Biochemistry methods - Carbohydrates 10058
Biochemistry studies - Proteins, peptides and amino acids 10064
Biochemistry studies - Carbohydrates 10068
Biophysics - Methods and techniques 10504
Biophysics - Molecular properties and macromolecules 10506
Biophysics - Membrane phenomena 10508
Blood - Blood cell studies 15004
Blood - Lymphatic tissue and reticuloendothelial system 15008
Bones, joints, fasciae, connective and adipose tissue - Anatomy 18002
Integumentary system - Anatomy 18502
Development and Embryology - Morphogenesis 25508
In vitro cellular and subcellular studies 32600
Immunology - Immunopathology, tissue immunology 34508
IT Major Concepts
Biochemistry and Molecular Biophysics; Blood and Lymphatics (Transport

and Circulation); Cell Biology; Clinical Endocrinology (Human Medicine, Medical Sciences); Development; Genetics; Integumentary System (Chemical Coordination and Homeostasis); Membranes (Cell Biology); Skeletal System (Movement and Support)

IT Miscellaneous Descriptors

ADHESION MOLECULES; ANTIGEN-PRESENTING CELL; T-HELPER CELL

ORGN Classifier

Hominidae 86215

Super Taxa

Primates; Mammalia; Vertebrata; Chordata; Animalia

Organism Name

human

Taxa Notes

Animals, Chordates, Humans, Mammals, Primates, Vertebrates

and Circulation); Cell Biology; Clinical Endocrinology (Human Medicine, Medical Sciences); Development; Genetics; Integumentary System (Chemical Coordination and Homeostasis); Membranes (Cell Biology); Skeletal System (Movement and Support)

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ADHESION MOLECULES; ANTIGEN-PRESENTING CELL; T-HELPER CELL

ORGN Classifier
Hominidae 86215
Super Taxa
Primates; Mammalia; Vertebrata; Chordata; Animalia
Organism Name
human
Taxa Notes
Animals, Chordates, Humans, Mammals, Primates, Vertebrates

ANSWER 24 OF 25 CAPLUS COPYRIGHT 2007 ACS on STN
AN 1996:54899 CAPLUS
DN 124:114965
ED Entered STN: 26 Jan 1996
TI B cell-B cell interaction through intercellular adhesion molecule-1 and lymphocyte functional antigen-1 regulates immunoglobulin E synthesis by B cells stimulated with interleukin-4 and anti-CD40 antibody
AU Katada, Yoshinori; Tanaka, Toshio; Ochi, Hiroshi; Aitani, Masakazu; Yokota, Akira; Kikutani, Hitoshi; Suemura, Masaki; Kishimoto, Tadamitsu
CS Department Medicine III, Osaka University Medical School, Osaka, Japan
SO European Journal of Immunology (1996), 26(1), 192-200
CODEN: EJIMAF; ISSN: 0014-2980
PB VCH
DT Journal
LA English
CC 15-3 (Immunochemistry)
AB IgE synthesis by purified human B cells is induced by two signals: a class switching factor, most commonly interleukin (IL)-4, and the engagement of CD40, which is activated through its interaction with CD40 ligand (CD40L) expressed on activated T cells. Thus, the combination of IL-4 and anti-CD40 monoclonal antibody (mAb) has been shown to stimulate IgE production in vitro by highly purified B cells. In this T cell-independent system, strong homotypic aggregation of B cells is observed prior to the production of IgE. Flow cytometric anal. and cell binding assays showed that the stimulation of purified B cells with anti-CD40 mAb plus IL-4 resulted in a striking increase of intercellular adhesion mol. (ICAM)-1(CD54) expression, an induction of CD43 and an avidity change of lymphocyte functional antigen (LFA)-1(CD11a/CD18), with little augmentation of CD18 expression. Addition of anti-ICAM-1 mAb caused an inhibition of homotypic aggregation but augmented IgE synthesis by B cells stimulated with anti-CD40 mAb and IL-4, although it did not affect B cell proliferation or IL-6 production by the B cells. Among the mAb against counter-receptors for ICAM-1 tested, anti-CD11a mAb suppressed IgE synthesis, while anti-CD18 mAb and anti-CD43 mAb had little effect. The enhancing or inhibitory effect of anti-ICAM-1 mAb or anti-CD11a mAb on IgE production was achieved by the increased or decreased expression of germline Cε transcripts by B cells stimulated with anti-CD4 mAb and IL-4. These results indicate that B cell-B cell interaction through ICAM-1 and one of its counter receptors, LFA-1, regulates IgE synthesis by modulating Cε germ-line transcription.
ST B cell IgE formation interleukin CD40; ICAM 1 LFA 1 IgE formation
IT Transcription, genetic
(B cell interaction through ICAM-1 and LFA-1 regulates IgE synthesis stimulated with interleukin-4 and anti-CD40 antibody)
IT Lymphocyte
(B-cell, B cell interaction through ICAM-1 and LFA-1 regulates IgE synthesis stimulated with interleukin-4 and anti-CD40 antibody)
IT Antigens
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); BIOL (Biological study)
(CD40, B cell interaction through ICAM-1 and LFA-1 regulates IgE synthesis stimulated with interleukin-4 and anti-CD40 antibody)
IT Immunoglobulins
RL: BSU (Biological study, unclassified); MFM (Metabolic formation); BIOL (Biological study); FORM (Formation, nonpreparative)
(E, B cell interaction through ICAM-1 and LFA-1 regulates IgE synthesis stimulated with interleukin-4 and anti-CD40

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DN 124:114965
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TI B cell-B cell interaction through intercellular adhesion molecule-1 and lymphocyte functional antigen-1 regulates immunoglobulin E synthesis by B cells stimulated with interleukin-4 and anti-CD40 antibody
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CS Department Medicine III, Osaka University Medical School, Osaka, Japan
SO European Journal of Immunology (1996), 26(1), 192-200
CODEN: EJIMAF; ISSN: 0014-2980
PB VCH
DT Journal
LA English
CC 15-3 (Immunochemistry)
AB IgE synthesis by purified human B cells is induced by two signals: a class switching factor, most commonly interleukin (IL)-4, and the engagement of CD40, which is activated through its interaction with CD40 ligand (CD40L) expressed on activated T cells. Thus, the combination of IL-4 and anti-CD40 monoclonal antibody (mAb) has been shown to stimulate IgE production in vitro by highly purified B cells. In this T cell-independent system, strong homotypic aggregation of B cells is observed prior to the production of IgE. Flow cytometric anal. and cell binding assays showed that the stimulation of purified B cells with anti-CD40 mAb plus IL-4 resulted in a striking increase of intercellular adhesion mol. (ICAM)-1(CD54) expression, an induction of CD43 and an avidity change of lymphocyte functional antigen (LFA)-1(CD11a/CD18), with little augmentation of CD18 expression. Addition of anti-ICAM-1 mAb caused an inhibition of homotypic aggregation but augmented IgE synthesis by B cells stimulated with anti-CD40 mAb and IL-4, although it did not affect B cell proliferation or IL-6 production by the B cells. Among the mAb against counter-receptors for ICAM-1 tested, anti-CD11a mAb suppressed IgE synthesis, while anti-CD18 mAb and anti-CD43 mAb had little effect. The enhancing or inhibitory effect of anti-ICAM-1 mAb or anti-CD11a mAb on IgE production was achieved by the increased or decreased expression of germline Cε transcripts by B cells stimulated with anti-CD4 mAb and IL-4. These results indicate that B cell-B cell interaction through ICAM-1 and one of its counter receptors, LFA-1, regulates IgE synthesis by modulating Cε germ-line transcription.
ST B cell IgE formation interleukin CD40; ICAM 1 LFA 1 IgE formation
IT Transcription, genetic
(B cell interaction through ICAM-1 and LFA-1 regulates IgE synthesis stimulated with interleukin-4 and anti-CD40 antibody)
IT Lymphocyte
(B-cell, B cell interaction through ICAM-1 and LFA-1 regulates IgE synthesis stimulated with interleukin-4 and anti-CD40 antibody)
IT Antigens
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); BIOL (Biological study)
(CD40, B cell interaction through ICAM-1 and LFA-1 regulates IgE synthesis stimulated with interleukin-4 and anti-CD40 antibody)
IT Immunoglobulins
RL: BSU (Biological study, unclassified); MFM (Metabolic formation); BIOL (Biological study); FORM (Formation, nonpreparative)
(E, B cell interaction through ICAM-1 and LFA-1 regulates IgE synthesis stimulated with interleukin-4 and anti-CD40

antibody)

IT Glycoproteins, specific or class
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); BIOL (Biological study)
(ICAM-1 (intercellular adhesion mol. 1), B cell interaction through ICAM-1 and LFA-1 regulates IgE synthesis stimulated with interleukin-4 and anti-CD40 antibody)

IT Integrins
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); BIOL (Biological study)
(antigens LFA-1, B cell interaction through ICAM-1 and LFA-1 regulates IgE synthesis stimulated with interleukin-4 and anti-CD40 antibody)

IT Lymphokines and Cytokines
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); BIOL (Biological study)
(interleukin 4, B cell interaction through ICAM-1 and LFA-1 regulates IgE synthesis stimulated with interleukin-4 and anti-CD40 antibody)

IT Sialoglycoproteins
RL: BSU (Biological study, unclassified); MFM (Metabolic formation); BIOL (Biological study); FORM (Formation, nonpreparative)
(leukosialins, B cell interaction through ICAM-1 and LFA-1 regulates IgE synthesis stimulated with interleukin-4 and anti-CD40 antibody)

antibody)

IT Glycoproteins, specific or class
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); BIOL (Biological study)
(ICAM-1 (intercellular adhesion mol. 1), B cell interaction through ICAM-1 and LFA-1 regulates IgE synthesis stimulated with interleukin-4 and anti-CD40 antibody)

IT Integrins
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); BIOL (Biological study)
(antigens LFA-1, B cell interaction through ICAM-1 and LFA-1 regulates IgE synthesis stimulated with interleukin-4 and anti-CD40 antibody)

IT Lymphokines and Cytokines
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); BIOL (Biological study)
(interleukin 4, B cell interaction through ICAM-1 and LFA-1 regulates IgE synthesis stimulated with interleukin-4 and anti-CD40 antibody)

IT Sialoglycoproteins
RL: BSU (Biological study, unclassified); MFM (Metabolic formation); BIOL (Biological study); FORM (Formation, nonpreparative)
(leukosialins, B cell interaction through ICAM-1 and LFA-1 regulates IgE synthesis stimulated with interleukin-4 and anti-CD40 antibody)

ANSWER 22 OF 25 CAPLUS COPYRIGHT 2007 ACS on STN
AN 1998:566926 CAPLUS
DN 129:314876
ED Entered STN: 07 Sep 1998
TI Surface expression and release of soluble forms of CD8 and CD23 in CD40- and IL-4-activated mononuclear cells from patients with Graves' disease (GD)
AU Itoh, M.; Uchimura, K.; Hayakawa, N.; Makino, M.; Hayashi, R.; Nagata, M.; Kakizawa, H.; Nagasaka, A.; Sakamoto, H.; Kuzuya, H.
CS Department of Internal Medicine, School of Medicine, Fujita Health University, Aichi, 470-1192, Japan
SO Clinical and Experimental Immunology (1998), 113(2), 309-314
CODEN: CEXIAL; ISSN: 0009-9104
PB Blackwell Science Ltd.
DT Journal
LA English
CC 15-8 (Immunochemistry)
AB The authors investigated the effect of T cell-dependent B cell activation on the surface expression and release of the soluble forms of CD8 and CD23 by peripheral blood mononuclear cells (PBMC) obtained from patients with GD vs. patients with Hashimoto's thyroiditis, and normal controls. Incubating the PBMC with anti-CD40 MoAbs and IL-4 increased the soluble CD23 levels in cells from all three groups. An increase in the number of CD23+ cells was observed in the PBMC from the patients with GD but not in PBMC from Hashimoto's thyroiditis or controls. Less soluble CD8 was released from anti-CD40 antibody and IL-4-stimulated PBMC obtained from patients with GD relative to those from the controls. In addition, the number of CD8+ cells was significantly reduced in stimulated PBMC from the GD patients relative to those from controls. Incubation of PBMC with anti-CD40 antibody plus IL-4 did not affect the proportions of CD4+, CD20+, Fas+CD4+, and Fas+CD8+ cells. The addition of T3 to cultured PBMC from controls did not reproduce the changes in CD23+ and CD8+ cells noted in the samples from GD patients. Thus, T cell-dependent B cell activation, mediated by a CD40 pathway, may reduce the number of CD8+ cells, causing exacerbation of GD.
ST CD8 CD40 interleukin 4 Graves disease; CD23 CD40 interleukin 4 Graves disease
IT Cell activation
(B cell; soluble CD8 and CD23 expression in CD40- and IL-4-activated mononuclear cells from humans with Graves' disease)
IT Immunoglobulin receptors
RL: BOC (Biological occurrence); BSU (Biological study, unclassified); BIOL (Biological study); OCCU (Occurrence)
(IgE type II, soluble; soluble CD8 and CD23 expression in CD40- and IL-4-activated mononuclear cells from humans with Graves' disease)
IT B cell (lymphocyte)
(activation; soluble CD8 and CD23 expression in CD40- and IL-4-activated mononuclear cells from humans with Graves' disease)
IT Graves' disease
T cell (lymphocyte)
(soluble CD8 and CD23 expression in CD40- and IL-4-activated mononuclear cells from humans with Graves' disease)
IT CD40 (antigen)
Interleukin 4
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); BIOL (Biological study)
(soluble CD8 and CD23 expression in CD40- and IL-4-activated mononuclear cells from humans with Graves' disease)
IT CD8 (antigen)
RL: BOC (Biological occurrence); BSU (Biological study, unclassified); BIOL (Biological study); OCCU (Occurrence)
(soluble; soluble CD8 and CD23 expression in CD40- and IL-4-activated mononuclear cells from humans with Graves' disease)

ANSWER 22 OF 25 CAPLUS COPYRIGHT 2007 ACS on STN
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AU Itoh, M.; Uchimura, K.; Hayakawa, N.; Makino, M.; Hayashi, R.; Nagata, M.; Kakizawa, H.; Nagasaka, A.; Sakamoto, H.; Kuzuya, H.
CS Department of Internal Medicine, School of Medicine, Fujita Health University, Aichi, 470-1192, Japan
SO Clinical and Experimental Immunology (1998), 113(2), 309-314
CODEN: CEXIAL; ISSN: 0009-9104
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DT Journal
LA English
CC 15-8 (Immunochemistry)
AB The authors investigated the effect of T cell-dependent B cell activation on the surface expression and release of the soluble forms of CD8 and CD23 by peripheral blood mononuclear cells (PBMC) obtained from patients with GD vs. patients with Hashimoto's thyroiditis, and normal controls. Incubating the PBMC with anti-CD40 MoAbs and IL-4 increased the soluble CD23 levels in cells from all three groups. An increase in the number of CD23+ cells was observed in the PBMC from the patients with GD but not in PBMC from Hashimoto's thyroiditis or controls. Less soluble CD8 was released from anti-CD40 antibody and IL-4-stimulated PBMC obtained from patients with GD relative to those from the controls. In addition, the number of CD8+ cells was significantly reduced in stimulated PBMC from the GD patients relative to those from controls. Incubation of PBMC with anti-CD40 antibody plus IL-4 did not affect the proportions of CD4+, CD20+, Fas+CD4+, and Fas+CD8+ cells. The addition of T3 to cultured PBMC from controls did not reproduce the changes in CD23+ and CD8+ cells noted in the samples from GD patients. Thus, T cell-dependent B cell activation, mediated by a CD40 pathway, may reduce the number of CD8+ cells, causing exacerbation of GD.
ST CD8 CD40 interleukin 4 Graves disease; CD23 CD40 interleukin 4 Graves disease
IT Cell activation
(B cell; soluble CD8 and CD23 expression in CD40- and IL-4-activated mononuclear cells from humans with Graves' disease)
IT Immunoglobulin receptors
RL: BOC (Biological occurrence); BSU (Biological study, unclassified); BIOL (Biological study); OCCU (Occurrence)
(IgE type II, soluble; soluble CD8 and CD23 expression in CD40- and IL-4-activated mononuclear cells from humans with Graves' disease)
IT B cell (lymphocyte)
(activation; soluble CD8 and CD23 expression in CD40- and IL-4-activated mononuclear cells from humans with Graves' disease)
IT Graves' disease
T cell (lymphocyte)
(soluble CD8 and CD23 expression in CD40- and IL-4-activated mononuclear cells from humans with Graves' disease)
IT CD40 (antigen)
Interleukin 4
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); BIOL (Biological study)
(soluble CD8 and CD23 expression in CD40- and IL-4-activated mononuclear cells from humans with Graves' disease)
IT CD8 (antigen)
RL: BOC (Biological occurrence); BSU (Biological study, unclassified); BIOL (Biological study); OCCU (Occurrence)
(soluble; soluble CD8 and CD23 expression in CD40- and IL-4-activated mononuclear cells from humans with Graves' disease)

RE.CNT 38 THERE ARE 38 CITED REFERENCES AVAILABLE FOR THIS RECORD
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AN 1994:178029 BIOSIS

DN PREV199497191029

TI Activated CD4+ T cells induce CD40-dependent proliferation of human B cell precursors.

AU Renard, Nathalie [Reprint author]; Duvert, Valerie; Blanchard, Dominique; Banchereau, Jacques; Saeland, Sem

CS Schering-Plough, Lab. Immunological Res., 27 chemin des Peupliers, 69571 Dardilly, France

SO Journal of Immunology, (1994) Vol. 152, No. 4, pp. 1693-1701. CODEN: JOIMA3. ISSN: 0022-1767.

DT Article

LA English

ED Entered STN: 26 Apr 1994
Last Updated on STN: 26 Apr 1994

AB Anti-CD3-activated human CD4+ T cell clones were found to induce proliferation of CD10+, CD19+, surface(s) Ig-B cell precursors (BCP) isolated from human fetal bone marrow. The great majority of the B lineage cells recovered in cocultures of BCP and activated T cells displayed a BCP phenotype (Ig- or cytoplasmic mu+ and K/lambda-), including most of the cycling cells, indicating that the cultures do not favor a transition to mature B cells. Supernatants of activated T cells were ineffective in inducing BCP proliferation, indicating the necessity of close association with stimulator cells. In line with this finding, the CD40 molecule was found to represent an important component of the cocultures, as BCP proliferation was strongly inhibited by soluble anti-CD40 antibody. In addition, CD4 + T cell clones from a hyper-IgM patient expressing a truncated CD40 ligand (CD40-L) failed to induce BCP proliferation. Finally, a combination of cytokines (IL-2, IL-3, IL-7, and IL-10) enhanced the observed T cell-dependent BCP proliferation, but could not substitute for the deficient CD40-L. Taken together, our data demonstrate that CD4+ T cells exert a stimulatory effect on in vitro B human lymphopoiesis via the CD40 pathway. The present results suggest that T cells may play an important role in regulating B cell ontogeny in the bone marrow.

CC Microscopy - Cytology and cytochemistry 01054
Cytology - Human 02508
Genetics - Human 03508
Radiation biology - Radiation and isotope techniques 06504
Biochemistry studies - Proteins, peptides and amino acids 10064
Biochemistry studies - Carbohydrates 10068
Biophysics - Molecular properties and macromolecules 10506
Biophysics - Membrane phenomena 10508
Blood - Blood cell studies 15004
Blood - Lymphatic tissue and reticuloendothelial system 15008
Bones, joints, fasciae, connective and adipose tissue - General and methods 18001
Bones, joints, fasciae, connective and adipose tissue - Anatomy 18002
Development and Embryology - Morphogenesis 25508
In vitro cellular and subcellular studies 32600
Immunology - General and methods 34502
Immunology - Immunopathology, tissue immunology 34508

IT Major Concepts
Biochemistry and Molecular Biophysics; Blood and Lymphatics (Transport and Circulation); Cell Biology; Clinical Endocrinology (Human Medicine, Medical Sciences); Development; Genetics; Immune System (Chemical Coordination and Homeostasis); Membranes (Cell Biology); Skeletal System (Movement and Support)

IT Miscellaneous Descriptors
B-CELL ONTOGENY; B-LYMPHOPOIESIS; BONE MARROW; CYTOFLUOROMETRY

ORGN Classifier
Hominidae 86215

ANSWER 14 OF 25 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN

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IT Major Concepts
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IT Miscellaneous Descriptors
B-CELL ONTOGENY; B-LYMPHOPOIESIS; BONE MARROW; CYTOFLUOROMETRY

ORGN Classifier
Hominidae 86215

Super Taxa
Primates; Mammalia; Vertebrata; Chordata; Animalia
Organism Name
Hominidae
Taxa Notes
Animals, Chordates, Humans, Mammals, Primates, Vertebrates

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Organism Name
   Hominidaé
Taxa Notes
   Animals, Chordates, Humans, Mammals, Primates, Vertebrates

L15 ANSWER 11 OF 25 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN
AN 2000:432374 BIOSIS
DN PREV200000432374
TI Increased expression of CD40 ligand in activated CD4+ T lymphocytes of systemic sclerosis patients.
AU Valentini, Gabriele [Reprint author]; Romano, Maria Fiammetta; Naclerio, Caterina; Bisogni, Rita; Lamberti, Annalisa; Turco, Maria Caterina; Venuta, Salvatore
CS Istituto di Clinica Medica e Reumatologia, II Universita di Napoli, Via Pansini, 5, 80131, Napoli, Italy
SO Journal of Autoimmunity, (August, 2000) Vol. 15, No. 1, pp. 61-66. print.
ISSN: 0896-8411.
DT Article
LA English
ED Entered STN: 11 Oct 2000
Last Updated on STN: 10 Jan 2002
AB CD40-CD154 interactions play a key role in regulating immune response and are involved in the development of some autoimmune diseases. We analysed the expression of CD154 antigen in CD3-activated PBMC from 10 systemic sclerosis (SSc) patients and 10 control subjects by immunofluorescence. PBMC from SSc patients showed an increased expression of this molecule, since, 6 h following CD3 stimulation, the percentage of CD154+ cells was of 17.53+-2.0 (mean+-SE) in control and 25.33+-2.93 in patient cells ($P<0.03$). The higher expression of CD154 antigen was ascribible to CD4+ cells. The enhanced induction of CD154 following CD3 stimulation depended on protein synthesis, since was abolished when the cells were stimulated via CD3 in the presence of cycloheximide. By analysing the expression of the CD40-induced antigen CD80, we verified that a blocking anti-CD40 antibody inhibited CD80 appearance in SSc activated monocytes, indicating that CD154 molecule was functional. These results show an enhanced expression of a functional CD154 molecule in SSc CD4+ activated T lymphocytes.
CC Cytology - Human 02508
Cytology - Animal 02506
Biochemistry studies - General 10060
Biochemistry studies - Proteins, peptides and amino acids 10064
Blood - Blood and lymph studies 15002
Blood - Blood cell studies 15004
Bones, joints, fasciae, connective and adipose tissue - Pathology 18006
Immunology - General and methods 34502
Immunology - Immunopathology, tissue immunology 34508
IT Major Concepts
Biochemistry and Molecular Biophysics; Clinical Immunology (Human Medicine, Medical Sciences)
IT Parts, Structures, & Systems of Organisms
CD154-positive cells: blood and lymphatics, immune system; CD4 -positive T lymphocytes: blood and lymphatics, immune system; PBMC: blood and lymphatics, immune system, CD3-activated
IT Diseases
systemic sclerosis: connective tissue disease
Sclerosis (MeSH)
IT Chemicals & Biochemicals
CD154: expression, functionality; CD3; CD80: CD40-induced antigen, expression; anti-CD40 antibody; cycloheximide: protein synthesis inhibitor; protein: synthesis
ORGN Classifier
Hominidae 86215
Super Taxa
Primates; Mammalia; Vertebrata; Chordata; Animalia
Organism Name

human: patient
Taxa Notes
Animals, Chordates, Humans, Mammals, Primates, Vertebrates
RN 66-81-9 (cycloheximide)

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FILE 'BIOSIS, CAPLUS, EMBASE, MEDLINE, JAPIO' ENTERED AT 15:23:46 ON 13 AUG 2007

L1	1924	S	(ANTI CD4 ANTIBOD?)
L2	839	S	(ANTI CD40 ANTIBOD?)
L3	3	S	L1 AND L2
L4	3		DUPLICATE REMOVE L3 (0 DUPLICATES REMOVED)
L5	0	S	(CDLOCD40HI)
L6	6661	S	CD4 AND CD40
L7	2554	S	L6 AND ANTIBOD?
L8	1638	S	L7 AND PD<2004
L9	876		DUPLICATE REMOVE L8 (762 DUPLICATES REMOVED)
L10	34	S	L9 AND DIABETES?
L11	1	S	L9 AND EMPHYSEMA?
L12	803		DUPLICATE REMOVE L1 (1121 DUPLICATES REMOVED)
L13	661	S	L12 AND PD<2004
L14	7	S	L9 AND L1
L15	25	S	L9 AND L2

LYcook 8/13/07

PALM INTRANET

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Biotech Query for 10/563570

Title: **METHODS FOR PREDICTING DEVELOPMENT OF AUTO-IMMUNE DISEASES AND TREATMENT OF SAME**

Inventor: **WAGNER, DAVID**

Location:

Location Date: Group Art Unit: **1641**

Status: **71/RESPONSE TO NON-FINAL OFFICE ACTION ENTERED AND FORWARDED TO EXAMINER**

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